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Effects of Strength and Endurance Exercise Order on Endocrine Responses to Concurrent Training

Submission type: Original Investigation

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Abstract

Purpose: The present study examined the effect of strength and endurance training order on the endocrine milieu associated with strength development and performance during concurrent training. **Methods:** A randomised, between groups design was employed with thirty recreationally resistance-trained males completing one of four acute experimental training protocols; strength training (ST), strength followed by endurance training (ST-END), endurance followed by either strength training (END-ST) or no training (CON). Blood samples were taken before each respective exercise protocol, immediately upon cessation of exercise, and 1 h post cessation of exercise. Blood samples were subsequently analysed for total testosterone, cortisol and lactate concentrations. **Results:** Ability to maintain 80% 1RM during strength training was better in ST and ST-END than END-ST (both $p < 0.05$). Immediately following the respective exercise protocols all training interventions elicited significant increases in testosterone ($p < 0.05$). ST and END-ST resulted in greater increases in cortisol than ST-END (both $p < 0.05$). The testosterone:cortisol ratio was similar following the respective exercise protocols. Blood lactate concentrations post-training were greater following END-ST and ST than ST-END (both $p < 0.05$). **Conclusions:** Conducting endurance exercise prior to strength training resulted in impaired strength training performance. Blood cortisol and lactate concentrations were greater when endurance training was conducted prior to strength training than *vice versa*. As such, it may be suggested that conducting endurance prior to strength training may result in acute unfavourable responses to strength training when strength training is conducted with high loads.

Key words: Training order, testosterone, cortisol, combined exercise and exercise sequence

Introduction

Whilst it appears both anabolic and catabolic endocrine factors are influenced by concurrent training (Bell, Syrotuik, Martin, Burnham, & Quinney, 2000; Bell, Syrotuik, Socha, Maclean, & Quinney, 1997; Kraemer et al., 1995), the acute responses of ‘primary’ hormones such as testosterone and cortisol in response to concurrent training are yet to be fully elucidated. Previous research has indicated the responses of testosterone and cortisol may be influenced by the order in which strength and endurance training are performed (Cadore, Izquierdo, dos Santos, et al., 2012). Moreover, inappropriate scheduling of strength and endurance training has previously been implicated in muted strength developed (García-Pallarés & Izquierdo, 2011; García-Pallarés, Sánchez-Medina, Carrasco, Díaz, & Izquierdo, 2009). This highlights the need for a greater understanding of the physiological responses to sequencing of strength and endurance training.

Various sequencing protocols have been employed within concurrent training paradigms, including; endurance before strength training on separate days (Sale, MacDougall, Jacobs, & Garner, 1990), strength before endurance training on separate days (Häkkinen et al., 2003; Hortobagyi, Katch, & Lachance, 1991), strength and endurance training on the same day but separate sessions (Craig, Lucas, Pohlman, & Stelling, 1991) and strength before endurance training (or *vice versa*) in the same session (Cadore et al., 2012; Chtara et al., 2005; Chtara et al., 2008). As such, the “optimal” sequence of strength and endurance training remains unclear.

Studies that have investigated the effects of intra-session sequencing of concurrent training on strength development have yielded equivocal findings. Previous research has demonstrated strength development is greater when strength precedes endurance training

rather than *vice versa* (Cadore, Izquierdo, Alberton, et al., 2012; Collins & Snow, 1993). In contrast, others have reported no differences in strength development following either sequence of strength and endurance training (Chtara et al., 2008; Davitt, Pellegrino, Schanzer, Tjionas, & Arent, 2014; Eklund et al., 2015; Eklund et al., 2015; Schumann et al., 2014; Rosa et al., 2015). The variances in findings are likely attributable to the differing strength training protocols employed, as it is widely accepted that the set x rep scheme and load imposed influences the responses of primary hormones (Spiering et al., 2008). In addition the training status of participants may have contributed to the variance in findings.

Whilst the aforementioned research provides information relating to the performance responses to concurrent training protocols, more recent research has examined the influence of intra- and inter-session sequencing of strength and endurance training on primary hormones; including testosterone and cortisol. It has been reported elevations in testosterone may be greater when strength loading is conducted following endurance loading, compared with strength prior to endurance loadings (Cadore et al., 2012; Schumann et al., 2014). The cortisol response to concurrent training has been observed to be greater when strength is conducted prior to endurance loading compared to *vice versa* (Taipale & Häkkinen, 2013), whereas others have reported similar testosterone and cortisol responses to either sequence of combined strength and endurance loadings (Eklund et al., 2015). As such, it appears that an “order effect” of strength and endurance loading does exist. It should also be noted that many studies in which the order effect of strength and endurance training has been investigated have employed differing participant groups, these include; recreationally trained (Eklund et al., 2015; Eklund et al., 2016) and inactive (Davitt et al., 2014) females, recreationally trained males (Rosa et al., 2015; Schumann et al., 2014), and combined male and female samples (Taipale & Häkkinen, 2013; Taipale et al., 2014). Much previous work has indicated that the

endocrine responses to exercise (particularly strength training) stimuli are highly dependent on maturity status, training status and sex (Kraemer et al., 1999; Kraemer & Ratamess, 2005; Spiering et al., 2008). As such it is presently difficult to make general conclusions about the influence of manipulating the order of strength and endurance training on endocrine factors.

Previous research has indicated that endocrine responses to concurrent training interventions may contribute to the inhibition of strength development (Bell et al., 2000; Bell et al., 1997; Kraemer et al., 1995). In addition, longer concurrent training interventions have been reported to elevate cortisol and possibly contribute attenuated fibre hypertrophy when compared with strength training alone (Kraemer et al., 1995). In addition, some studies in which elevated cortisol has been reported have also observed attenuated strength development following concurrent training strategies (Bell et al., 2000; Kraemer et al., 1995). Based on these findings it is reasonable to suggest that the relative responses and ratios of testosterone and cortisol could shift the endocrine system in favour of a catabolic state following concurrent training (Bell et al., 2000; Bell et al., 1997; Kraemer et al., 1995; Kraemer & Ratamess, 2005). Additionally, it remains speculative whether an acute bout of concurrent loading can elicit similar anabolic responses to strength loading alone if the sequence of exercise is appropriately programmed.

The purpose of the present study was to investigate the effects of manipulating the order of strength and endurance loadings on strength training performance and endocrine factors which may impact upon strength training adaptation in recreationally trained men.

Methods

Subjects

Thirty healthy recreationally resistance-trained men (age: 24 ± 4 y; body mass: 80.0 ± 9.0 kg; stature: 179.8 ± 6.8 cm; % body fat: 15.1 ± 5.3 %; sum of assessed (1 repetition maximum) 1RMs: 444.0 ± 50.0 kg; $\text{VO}_{2\text{max}}$: 50.0 ± 6.3 ml·kg·min) volunteered to participate in the study. Participants were matched at baseline for age, body mass, total of 1 RMs (sum of load achieved in back squat, bench press, bent over row, military press and deadlift) (all $p > 0.05$) then randomly assigned to a specific experimental condition. Each participant had completed > 2 years of strength training prior to the study. All participants were free from any endocrine or metabolic contraindications and in all cases participants were asked to refrain from nutritional supplementation or pharmacological interventions for 30 days prior to testing. The University's Ethics Committee, in accordance with the Declaration of Helsinki, ratified all experimental procedures; subsequent written informed consent was then attained from all volunteers.

Design

A randomised, between-group study design was employed. Participants were randomly assigned to one of 4 experimental conditions: i) strength loading (ST) ($n = 8$), ii) concurrent training with strength loading conducted first (ST-END) ($n = 8$), iii) concurrent training with endurance loading first (END-ST) ($n = 8$) or iv) no loading (CON) ($n = 6$). A schematic of the experiment is presented in Figure 1.

Prior to commencing any trials, assessment of $\text{VO}_{2\text{max}}$ (via an incremental treadmill protocol) and 1RM loads were performed for the purpose of normalising relative loading intensity, all assessments were conducted in line with standardised procedures (Rønnestad, Hansen, &

Raastad, 2011; Walshe et al., 2010). To assess the effect of each intervention on endocrine factors relating to strength and morphological adaptation, venous blood samples were taken and subsequently analysed for total testosterone, cortisol and lactate. Samples were taken immediately preceding (pre), immediately following (post), and 1 h post cessation of the strength training protocol. To determine if the sequence of strength and endurance training performed affected strength training performance, participant's ability to maintain their predetermined load during the strength training protocol was assessed via recording the load as % 1RM during the experimental protocol. In the CON condition venous blood samples were drawn at rest, ~1 h later as this was the time it typically took participants to complete the strength loading protocol and 1 h after. This mimicked the timings of blood draws in the training conditions.

Figure 1 about here

Methodology

The strength loading consisted of the back squat, bench press, bent over row, military press and deadlift. Exercises were selected as they are compound movements that involve the major joints and muscle groups of the body and reflect exercises commonly used as part of a holistic strength training strategy. For each exercise 5 sets of 6 repetitions at 80% 1RM were completed, with 2 min rest intervals between sets. If participants were unable to maintain 80% 1RM, the load was adjusted to ensure 5 sets of 6 repetitions could be completed. Strength training protocols of this nature which stimulate large muscle mass and involve shorter rest periods elicit large increases in the endocrine factors assessed within this study (Kraemer et al., 2008; Volek, Kraemer, Bush, Incledon, & Boetes, 1997). In all instances, the endurance loading protocol required participants to run on a treadmill (hp Cosmos, Pulsar,

Nussdorf-Traunstein, Germany) at their pre-determined running velocity of 70% of at $\text{VO}_{2\text{max}}$ for 30 min.

All strength and endurance exercise commenced at the same time of day (10:00 h \pm 1 h) to avoid any diurnal performance or endocrine variations (Hayes, Bickerstaff, & Baker, 2010). Participants arrived at the lab having refrained from consuming food or caffeine for 2 h prior to assessment. Participants were also advised to abstain from general exercise for 24 h pre visit and strength training 5 days pre visit.

Venous blood samples were collected from the antecubital fossa. Whole blood was centrifuged (accuSpin 3R, Fisher Scientific, Loughborough, UK) at 4°C at 1509 g for 10 min, after which, the plasma was aspirated, aliquoted and immediately stored at -80°C. Blood samples were analysed for testosterone, cortisol and lactate (Lac^-) concentrations. Analysis of Lac^- were performed using a desk top device (Biosen C_Line Sport, EKF Diagnostic, Barleben, Germany), with a detect range of 0.5 – 40.0 mMol/L⁻¹.

Plasma testosterone and cortisol were measured in duplicate via commercially available enzyme-linked immunosorbent assay (ELISA) kits (IBL International, Hamburg, Germany) and in accordance with the manufacturer's instructions. For testosterone there was a minimum detection limit of 0.2 nMol·L⁻¹, inter-assay and intra-assay variation of 4.2 – 7.4 and 3.1 – 5.4 and the calibration curve revealed Pearson's correlation coefficients (r) = 0.99. For cortisol there was a minimum detection limit of 6.8 nMol·L⁻¹ with an inter-assay and intra-assay variation of 2.1 – 5.0 and 2.6 – 3.5, the calibration curve revealed r = 0.99, respectively.

Statistical Analysis

Data are presented as mean \pm standard deviation and the alpha level of 0.05 was set prior to data analysis. Prior to analysis dependant variables were verified as meeting required assumptions of parametric statistics. Assumptions of sphericity were assessed using Mauchly's test of sphericity, if the assumption of sphericity was violated Greenhouse Gessier correction was employed. ANOVA analysed differences between 4 conditions (ST, ST-END, END-ST and CON) and 3 time points (pre, immediately post and 1 h post exercise cessation). Values of testosterone and cortisol were transformed to percentage change ($\Delta\%$) from baseline for the purpose of analysis. Changes in testosterone, cortisol and Lac^- were analysed using mixed model repeated measures ANOVA tests. Participant's ability to maintain their individual required training intensity and RPE were analysed using one-way ANOVA. The ANOVA analysed differences between 3 conditions ST, ST-END and END-ST). If significant effects between conditions or over time were observed *post-hoc* differences were analysed via Bonferroni correction. Statistical power of the study was calculated post-hoc using G*Power statistical software (v3.1.3, Düsseldorf, Germany) using the effect size, group mean, SD and sample size of the primary outcome measures, in this case being strength training performance and endocrine factors. Power was calculated as between 0.8 and 1 indicating sufficient statistical power (Cohen, 1992).

Results

The ability to maintain required relative strength loading intensity was different between groups ($F_{(2, 20)} = 11.25$, $p = 0.001$). The ST group were able to maintain relative strength loading intensity $4.7 \pm 1.7 \%$ ($p = 0.007$; Figure 2) better than END-ST ($70.1 \pm 3.9 \%$ 1RM). ST-END also resulted in participants achieving a significantly higher ($p < 0.001$) relative

strength loading intensity than END–ST ($7.6 \pm 1.7 \%$), with no differences between ST and ST-END.

Figure 2 about here

A significant time x group interaction ($F_{(4, 34)} = 5.577, p = 0.001$) and a time effect ($F_{(1, 34)} = 58.230, p < 0.001$) were observed for testosterone. Baseline testosterone levels were as follows; ST; 16.2 ± 4.9 nMol/L⁻¹, ST-END; 18.7 ± 8.1 nMol/L⁻¹, END-ST; 14.5 ± 2.7 nMol/L⁻¹ and CON; 16.1 ± 1.4 nMol/L⁻¹. All loading conditions elicited significant increases in testosterone immediately following exercise (ST; $44.1 \pm 23.2\%$, ES = 0.15, ST-END; $28.6 \pm 9.4\%$, ES = 0.56, END-ST; $36.1 \pm 23.5\%$, ES = 1.63 all $p < 0.001$) (Figure 3, Panel A). From immediately post-exercise to 1 h post exercise testosterone levels decreased significantly in all loading conditions (ST; $39.1 \pm 15.5\%$, ES = 0.10, $p < 0.001$, ST-END; $28.6 \pm 5.7\%$, ES = 0.37, $p = 0.01$, END-ST; $45.7 \pm 17.8\%$, ES = 2.24, $p < 0.001$). 1 h post resistance exercise cessation END–ST resulted in significantly lower testosterone levels than base ($9.6 \pm 5.8\%$, ES = 0.60, $p < 0.001$). 1 h post strength loading cessation END–ST resulted in $14.6 \pm 1.9\%$ and $13.3 \pm 9.3\%$ lower testosterone levels than ST and ST-END (Figure 3, Panel A) ($p < 0.001$ and $p = 0.02$ respectively). All loading conditions resulted in significantly greater post exercise increases in testosterone than CON (all $p < 0.001$).

Figure 3 about here

A significant time x group interaction and a time effect were also reported for cortisol ($F_{(5, 40)} = 3.553, p = 0.005$ and $F_{(2, 40)} = 33.051, p < 0.001$ respectively). Baseline cortisol levels were as follows; ST; 249.7 ± 107.2 nMol/L⁻¹, ST-END; 296.8 ± 93.9 nMol/L⁻¹, END-ST; $254.7 \pm$

50.4 nMol/L⁻¹ and CON; 291.6 ± 65.0 nMol/L⁻¹. Immediately post loading all conditions other than CON resulted in significant increases in cortisol (ST; 112.5 ± 52.4%, ES = 1.67, ST-END; 65.3 ± 34.3%, ES = 1.37, END-ST; 124.3 ± 73.1%, ES = 2.48, all $p < 0.001$) (Figure 3, Panel B). After 1 h post resistance exercise cessation cortisol levels decreased significantly in all training conditions (ST; -93.2 ± 14.3%, ES = 1.39, $p = 0.01$, ST-END; -52.3 ± 7.9%, ES = 1.09, $p = 0.01$, END-ST; -101.0 ± 32.6%, ES = 1.83, $p = 0.01$). Cortisol levels immediately post exercise increased significantly more in ST (47.2 ± 18.1%, $p = 0.03$) and END-ST (59.0 ± 38.8%, $p = 0.04$) than ST-END (Figure 3, Panel B).

Testosterone:cortisol ratio (T:C Ratio) was not significantly different between conditions ($F_{(3, 26)} = 5.665$, $p = 0.361$) nor over time ($F_{(1, 26)} = 2.442$, $p = 0.097$, Table 1).

Table 1 about here

A significant time x group interaction ($F_{(4, 34)} = 36.952$, $p < 0.001$) and an effect of time ($F_{(1, 34)} = 195.663$, $p < 0.001$) were observed for Lac⁻. All conditions other than CON elicited significant increases in Lac⁻ post loading (all $p < 0.001$). Significant decreases from post to 1 h post loading were also observed in all conditions, with the exception of CON (all $p < 0.001$). END-ST was the only condition in which Lac⁻ remaining significantly elevated from pre loading ($p = 0.001$).

ST resulted in 52.3 ± 84.6% and 49.5 ± 28.0% greater Lac⁻ post and 1 h post loading than ST-END ($p < 0.001$ and 0.002 respectively). END-ST also increased Lac⁻ 67.7 ± 5.6% and 56.9 ± 16.2% greater than ST-END post and 1 h post loading respectively (both $p < 0.001$).

Lac⁻ responses were significantly correlated with cortisol and testosterone responses to the respective loading protocols ($r = 0.57, p = 0.001$; $r = 0.68, p = 0.001$).

Discussion

The aim of the present study was to examine the influence of contrasting intra-session sequencing of strength and endurance loadings on endocrine factors, which might contribute to strength development and the interference phenomenon. It was observed that following the respective experimental protocols, strength following endurance loading resulted in greater elevations in cortisol and Lac⁻ than *vice versa*. All loading conditions resulted in elevated testosterone and cortisol immediately post loading. The large increases of testosterone and cortisol ($44.1 \pm 23.1\%$ and $124.3 \pm 71.3\%$, respectively in the ST condition) might be attributed to exercise selection, with the lifts involving large muscle mass (Kraemer et al., 2008; Volek et al., 1997).

Contrary to the findings of the present study testosterone has been reported to be greater when strength loading is performed after endurance rather than *vice versa* (Cadore et al., 2012; Rosa et al., 2015). This disparity in findings is likely due to the variances in experimental loading protocols employed in the respective studies. The strength loading protocol in the present study consisted of compound lifts stimulating large muscle masses which has been associated marked increases in testosterone (Kraemer et al., 1990). Following END-ST Rosa et al. (2015) observed a similar increases in testosterone to that of the present study as the authors also employed exercises such as the back squat, leg press and bench press involving large muscle masses. However it appears that like Cadore et al. (2012) when strength was followed by endurance loading the testosterone levels returned to baseline. As previously stated the strength loading protocol employed here stimulated large increases in

testosterone, it is likely that the magnitude of these increases were great enough to be maintained throughout and following the 30 min endurance stimulus. It is unclear as to why testosterone levels in END-ST were lower than baseline 1 h post loading. It may be suggested that the demanding strength training protocol resulted in high levels of stress and metabolic acidosis result in a slow decrease in testosterone levels. It may also speculated that the reduction in testosterone levels 1 h post END-ST are attributable to the negative feedback stimulus of the hypothalamic-pituitary-gonadal axis.

Similar to the findings of the present study others have reported no order effect on testosterone (Eklund et al., 2015; Schumann et al., 2014; Taipale & Häkkinen, 2013). In the case of Eklund et al. (2016) it is likely that the similar testosterone responses are attributable to the untrained (and female) nature of the study population. Previous research has indicated that training status may influence the endocrine responses to training (Ahtiainen, Pakarinen, Alen, Kraemer, & Häkkinen, 2003; Tremblay, Copeland, & Van Helder, 2004), and untrained individuals may exhibit a blunted endocrine response to strength training when compared with those who are strength trained (Tremblay et al., 2004). As all participants in the present study had 2 years of strength training history and as such were considered resistance trained it is unlikely that the training status of accounts for the similar testosterone responses reported here. Both Schumann et al. (2014) and Taipale and Häkkinen (2013) reported no order effect on testosterone responses, the authors also observed no significant increases in testosterone in either condition (ST-END or END-ST). As previously discussed programme variables such as load, set/rep scheme and rest periods influences the endocrine responses to strength loading (Häkkinen & Pakarinen, 1993. Schumann et al. (2014) and Taipale and Häkkinen (2013) both incorporated aspects of strength and power loadings (as opposed to hypertrophy),

this may explain the similar responses of testosterone to both sequences of strength and endurance loading and lack of significant elevations in testosterone.

Cortisol was significantly lower immediately post loading in the ST-END condition than END-ST (Figure 3, Panel B). This may indicate decreased catabolism when strength is conducted prior to endurance loading as opposed to *vice versa*. This hypothesis should be interpreted with caution as blood samples were only drawn post and 1 h post both bouts of exercise and not after each individual bout. Lac^- were significantly correlated with cortisol responses ($R = 0.57$), both lactate and cortisol have also been related to increased metabolic stress (Urhausen, Gabriel, & Kindermann, 1995). As such, it is possible this indicates the greater Lac^- and cortisol observed when endurance preceded strength exercise are reflective of greater metabolic stress when compared with ST-END. It is however possible that the proximity of blood sampling to the strength loading resulted in the greater cortisol response to ST and END-ST than ST-END. This is attributable to the demanding nature of the strength training protocol and the fact it elicited high levels of testosterone and cortisol. It is also possible that the 30 min endurance loading following the strength protocol allowed time for cortisol (and blood lactate) levels to decrease. Rosa et al. (2015) reported similar elevations in cortisol following both ST-END and END-ST. Whilst the participant sample was similar there are slight variances in both the endurance and strength loading protocols employed, this may account for the disparity in findings. In addition, Rosa et al. (2015) used a cross over design which resulted in the same participants completing both experimental conditions. The between groups design employed in the present study meant different participants completed the differing experimental conditions. This may be a limitation of the present study due to any individual hormonal responses to the loading stimuli imposed (Ahtiainen et al., 2003).

A potential limitation of the present study is that biochemical variables were not assessed during a more prolonged recovery period. Previous work has employed longer follow up analyses of 24 and 48 h post exercise (Schumann et al., 2014; Taipale & Häkkinen, 2013). This is particularly important in the concurrent training paradigm as much of the adaptation process occurs in the 48 h post any loading stimuli (Spiering et al., 2008). As such future studies in which mechanistic factors which may contribute to any interference characteristics are assessed where possible should perform analysis for prolonged recovery periods. An additional limitation of the present study is that androgen receptors and consequently the hormone-receptor interaction was not assessed.

Both ST and ST-END resulted in better performance during strength loading than END-ST (4.7 ± 0.6 and $7.6 \pm 2.4\%$ respectively). These data indicate strength performance and the ability to maintain a designated load is negatively affected by a preceding bout of endurance training, which is consistent with previous research (Sporer & Wenger, 2003). It is likely that this compromised strength performance as a result of prior endurance loading can be attributed to a greater build-up of inorganic phosphates demonstrated by the $67.7 \pm 5.6\%$ greater Lac^- post training in END-ST. If strength performance is repeatedly impaired as a result of preceding endurance training, the magnitude of strength training related adaptation may be attenuated compared to conducting strength training alone. This hypothesis has been supported by research demonstrating strength gains to be greater when strength training precedes endurance training or is performed in isolation (García-Pallarés et al., 2009). It is possible that the greater Lac^- following the END-ST loading was due to ST loading requiring more energy provided by the anaerobic glycolytic pathway than END loading. As such there may be other factors which contributed to the decreased strength performance following

END-ST. These may include; neural fatigue or muscular fatigue due to the eccentric loading associated with prolonged running.

This study sought to further elucidate the effects of acute sequencing of strength and endurance loadings on endocrine responses associated with strength training adaptation. The results of this study show that the manipulation of the order of acute strength and endurance loadings can influence the cortisol response without any concomitant changes in circulating testosterone. END-ST resulted in greater cortisol and Lac^- than ST-END. The present study's data also support the hypothesis that endurance immediately prior to strength loading reduces the quality of strength performance.

Due to logistical issues, at times it is inevitable that athletes and recreationally trained individuals will perform a combination of strength and endurance training in the same session or in close proximity. Performing endurance prior to strength training elicited greater increases in cortisol than *vice versa*, cortisol responses were however similar between END-ST and ST. It however is clear that prior endurance loading results in diminished quality of strength performance.

Conclusions

The underpinning mechanisms contributing to interference are unclear, previous research has indicate endocrine factors play some role in the attenuated strength development associated with concurrent training. The findings of the present study are insufficient to support this argument but do indicate that the order in which strength and endurance training are performed influence performance during strength training.

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Table 1. Effects of respective training interventions on testosterone:cortisol (T:C) ratio responses.

Condition	Stage		
	Pre	Post	1 h post
ST			
T:C Ratio (x10 ³)	97.2 ± 112.1	66.1 ± 71.9	136.3 ± 242.5
ST - END			
T:C Ratio (x10 ³)	65.6 ± 22.6	53.1 ± 18.3	63.4 ± 22.2
END – ST			
T:C Ratio (x10 ³)	59.6 ± 18.7	43.0 ± 33.5	44.6 ± 11.0
CON			
T:C Ratio (x10 ³)	58.1 ± 16.4	55.0 ± 9.0	58.5 ± 19.4

No statistically significant differences between conditions were observed at pre ($p = 0.194$).

Figure captions

Figure 1. Schematic representation of experimental time line. ST ($n = 8$) and ST - END ($n = 8$), END - ST ($n = 8$) and CON ($n = 6$).

Figure 2. Mean training intensity achieved in the ST ($n = 8$), ST-END ($n = 8$) and END-ST ($n = 8$) conditions. ST, strength loading alone; ST-END, strength loading followed by endurance loading; END-ST, endurance followed by strength loading. * Significantly greater than END-ST ($p = 0.007$). Dashed line indicates prescribed training intensity.

Figure 3. Mean relative testosterone (panel A) and cortisol (Panel B) responses the ST ($n = 8$), ST-END ($n = 8$), END-ST ($n = 8$) and CON ($n = 6$) conditions. ST, strength training alone; ST-END, strength loading followed by endurance loading; END-ST, endurance followed by strength loading; CON, no loading. Dashed line indicates zero. * Significantly greater than pre, in all loading conditions ($p < 0.001$), ** significantly lower than post in all loading conditions ($p < 0.01$), ‡ significantly greater increase in ST and ST-END than END-ST ($p < 0.05$), † ST and END-ST significantly greater than ST-END ($p < 0.05$).



